Topic 15 – Myocardial hypoxia, reperfusion, stroke – D

April 25th, Friday 2014

0227

Fragmented QRS in patients with STEMI undergoing PCI: relation to ST-segment resolution and determinism

Patrick Ohlmann (1), Celine Zerbib (1), Jean-Pierre Monassier (2), Bogdan Radulescu (1), Philippe Germain (1), Jérome Rischner (1), Philippe Reydel (3), Sebastien Hess (1), Nathan Messas (1), Morel Olivier (1)
(1) Hôpitaux Universitaires de Strasbourg, Pôle cardiovasculaire médico-chirurgical, Strasbourg, France – (2) Clinique du Diaconat, Mulhouse, France – (3) Hôpitaux Universitaires, SAMU, Strasbourg, France

Background: fragmented QRS complex (fQRS) is a marker of myocardial scarring and has prognostic significance. ST-segment resolution (STR) in ST-segment elevation acute myocardial infarction (STEMI) is characteristic of tissue reperfusion. This study investigates the relationship between fQRS and STR in STEMI patients undergoing primary percutaneous coronary intervention (PCI) and determines the predictors of fQRS.

Methods: we analyzed the electrocardiograms (ECGs) of 256 patients included in a multicenter prospective STEMI-PCI study. fQRS and ST-segment resolution were evaluated upon arrival in the ambulance (ECG-amb) and 1 h post-PCI (ECG-post). Major clinical cardiac events were assessed at 30 days and 6 months.

Results: fQRS was present in 33 patients (13.6%) on ECG-amb and in 30 patients (12%) on ECG-post. The presence of fQRS at either time was not associated with STR or clinical outcomes. In a multivariable analysis, the independent predictors of fQRS on ECG-amb were female sex (p=0.04), cardiac troponin Ic level at 72 h (p=0.01), TIMI 0-1 flow rate pre-PCI (p=0.002), and inferior STEMI location (p=0.04). Patients with fQRS on ECG-amb presented a larger necrosed mass on cardiac MRI than patients without fQRS (p=0.04). No predictors of fQRS post-PCI were identified.

Conclusion: the presence of fQRS at the time of presentation or 1 h after PCI was not associated with STR. However, fQRS was related to enzymatic infarct size, inferior STEMI location, and low TIMI flow rate.

0268

TREM-1 modulation improves cardiac function during myocardial infarction in pigs

Jeremie Lemarie (1), Amir Boufenzer (2), Marc Derive (2), Nguyen Tran (3), Sébastien Gibot (1)
(1)CHU Nancy, Réanimation Médicale, Nancy, France – (2) UMRS 1116, Vandoeuvre Les Nancy, France – (3) Ecole de Chirurgie, Vandoeuvre Les Nancy, France

Introduction: The widespread use of reperfusion therapy have led to an important improvement in short-term mortality after acute myocardial infarction (MI), but long-term mortality remains high. The Triggering Receptor Expressed on Myeloid cells-1 (TREM-1) belongs to the immunoreceptors superfamily and acts as an amplifier of the inflammatory response triggered by TLRs engagement during both infectious and aseptic inflammatory diseases. We hypothesized that administration of LR12, a synthetic peptide able to inhibit TREM-1 activation, could be beneficial at the acute phase of MI, in a clinically relevant model of experimental MI in pigs.

Materials and methods: MI was induced in fifteen anesthetized and mechanically ventilated pigs weighing 45-55 kg, by inflation of an angioplasty balloon in the proximal left anterior descending (LAD) coronary artery cannulated under X-ray guidance, during 60 minutes. Fifteen minutes before deflation, animals were randomized to receive either LR12 (n=7) or LR12-scramble (n=8). Complete hemodynamic and functional parameters were monitored through arterial line, swan-ganz and intraventricular conductance catheters. Resuscitation was conducted by experienced intensivists according to standard protocols used in clinical practice. The monitoring was prolonged until H18, then survivors were euthanized.

Results: The decrease in mean arterial pressure (MAP) was significantly limited during the monitoring period from H12 to the end (–22.1% vs –3.9%, p<.01). Cardiac index and cardiac power index, one of the strongest hemodynamic correlate of mortality in cardiogenic shock, were preserved under LR12 regimen (72% vs 45% from baseline value, p<.05) as well as SvO2 value (74% vs 62%, p<.05). Ejection fraction and parameters of systolic function improved under LR12 treatment.

Conclusion: TREM-1 inhibition by LR12 at the acute phase of myocardial infarction in invasively monitored pigs limits reperfusion injury and alteration of ventricular contractility.

0208

Mitochondrial-dependent cardioprotection during postischemic reperfusion is preserved in isolated aged rodent hearts

Oana Duicu (1), Andreea Privistirescu (1), Lavinia Noveanu (1), Denis Angoulvant (2), Istvan Baczko (1), Norbert Jost (1), Danina M. Muntean (1)
(1) "Victor Babeş" University of Medicine and Pharmacy, Pathophysiology, Timisoara, Roumanie – (2) CHRU Tours, Cardiologie USCI-Pôle Coeur Thorax Vaisseaux, Tours, France

It is widely accepted that with the demographic shift towards an ageing population prevalence of age-related cardiovascular disease will increase. Whether cardioprotective strategies are still effective in elderly is a matter of debate. We have previously demonstrated that magnesium orotate (MO) was cardioprotective in adult rat hearts subjected to acute ischemia/reperfusion (I/R) injury. The present study was purported to assess the persistence of the protective effects of MO in old Sprague-Dawley rats. To this aim isolated hearts from old (20-24 mo) vs. adult (4-6 mo) animals subjected to a protocol of 30 min I/120 min R were randomized to receive throughout the postischemic reperfusion: no intervention, MO, MgCl2 and orotic acid (OA). The effects on functional recovery and infarct size were compared to the ones elicited by CsA (0.2 μM), the classic mitochondrial permeability transition pore (mPTP) desensitizer. In a second group of experiments, cardiac mitochondria were isolated at 15 min of postischemic reperfusion for respiratory function and calcium retention capacity assessment. Acute administration of MO, MgCl2, but not OA (5 mM each) at the very onset of reperfusion was associated with a significant recovery of left ventricular developed pressure and infarct size reduction in both adult and old animals; protection was comparable to the one elicited by CsA. In mitochondria energized with complex I (but not complex II) substrates isolated from the old animals, all these pharmacological agents protected against the loss of outer mitochondrial membrane integrity, albeit in a lesser degree compared to adult mitochondria. MO, MgCl2, and CsA also prevented the calcium triggered-opening of the mPTP. In conclusion, magnesium containing-pharmacological agents as well as CsA were effective in protecting mitochondrial function at reperfusion in the aged rodent heart.

This work was supported by a grant of the Ministry of National Education.

0324

Cardioprotection against ischemia-reperfusion injury by heart rate control

Viviana Delgado Betancourt (1), Pietro Mesirca (1), Aurélie Covinhes (1), Isabelle Bidaud (1), Joël Nargeot (1), Christophe Piot (1), Jörg Striessnig (2), Matteo Mangoni (1), Stéphanie Barrère-Lemaire (1)
(1) Institut de Génomique Fonctionnelle, Cardioprotection, Montpellier, France – (2) Universität Innsbruck, Innsbruck, Autriche

Myocardial infarction (MI) is the major cause of cardiovascular mortality in western countries. Early reperfusion is the only treatment recommended to reduce infarct size (IS), a crucial prognostic factor of morbidity